Health Effects of Low-Level Hydrogen Sulfide in Ambient Air

(A Science Report to Governor John Engler)

Prepared by
Michigan Environmental Science Board
Hydrogen Sulfide Investigation Panel

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AUGUST 2000

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TABLE OF CONTENTS

Page

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PREFACE

Michigan Environmental Science Board

The Michigan Environmental Science Board (MESB) was created by Governor John Engler by Executive Order 1992-19 on August 6, 1992. The MESB is charged with advising the Governor, the Natural Resources Commission, the Michigan Department of Natural Resources and other state agencies, as directed by the Governor, on matters affecting the protection and management of Michigan's environment and natural resources. The MESB consists of nine members and an executive director, appointed by the Governor, who have expertise in one or more of the following areas: engineering, ecological sciences, economics, chemistry, physics, biological sciences, human medicine, statistics, risk assessment, geology and other disciplines as necessary. Upon the request of the Governor to review a particular issue, a panel, consisting of MESB members with relevant expertise, is convened to evaluate and provide recommendations on the issue. The MESB is neither a state policy body nor an advocate for or against any particular environmental or public health concern.

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Major Findings and Conclusions

Hydrogen sulfide (H_2S) is a colorless, highly toxic gas at high concentrations. It has a distinctive "rotten egg" odor and is produced by a variety of sources, including wastewater treatment plants, agricultural operations, paper mills and other manufacturing processes, and oil and gas development. Within Michigan, there is particular concern over H_2S associated with the exploration, production, and transportation of crude oil and natural gas. An extensive body of rules covering H_2S-containing wells, storage and treatment facilities, and pipelines was promulgated in Michigan in 1987. The rules prohibit nuisance odors from H_2S and prescribe installation of specific control equipment on H_2S-producing wells. Recently, however, public concern over H_2S associated with the oil and gas industry has escalated. In 1996, there was an intentional release (below worker safety standards) at a well in northern Michigan that affected 11 citizens in the area. Also, construction of a pipeline to transport H_2S-bearing natural gas from a well to a processing plant raised fears among citizens concerning the possibility of harm from accidental releases.

A considerable body of literature exists on the human health impacts resulting from exposure to high levels of H_2S. Death is often observed in accidental high dose exposures. In contrast, very little data have been compiled regarding health impacts from exposure to much lower levels of the gas that can be detected by its characteristic odor. On May 27, 1998, the Michigan Environmental Science Board (MESB) was charged by Governor John Engler to review the available H_2S low-level data and to recommend a range of exposures that could be considered safe to human health.

The MESB report presents background information on the properties, ambient concentrations, and atmospheric fate of H_2S. In addition, discussions are provided regarding the development of regulations, limitations associated with measurement of low level H_2S, and the current monitoring database for H_2S exposure in Michigan. Finally, an extensive review of the published scientific literature and results from on-going H_2S toxicological investigations is also provided. The major findings and conclusions of the MESB report are summarized below.

♦ Based on published scientific literature and results from on-going investigations reviewed in the report, the no observable adverse effect level (NOAEL) of H_2S in ambient air is between 2 and 10 parts per million (ppm). The lower value is from studies showing no increase in blood lactate in exercising adults. An exposure-related effect was observed after 5 ppm H_2S for 20 minutes. It represents a change that can result from inhibition of an enzyme (cytochrome oxidase) required for cellular respiration. Inhibition of that important enzyme is responsible for the acute toxic effects of H_2S. The higher value, 10 ppm, was the NOAEL found in a recent 70-day exposure study using laboratory animals (rats). A concentration of 30 ppm of the gas produced evidence of nasal lesions affecting olfactory nerve cells in that study. This is a definite toxic effect in a species that may be more sensitive than the human to nasal lesions from irritant gasses such as H_2S.

♦ NOAEL values obtained from experiments of high quality are usually divided by safety factors to produce a value representing a safe concentration of a substance in the air. This increases the certainty that humans exhibiting high sensitivity to the toxic effects of the gas will be protected. These safety factors should be applied by governmental agencies using knowledge of the characteristics of the toxicity of H_2S gained from human and laboratory animal experiments. Selection of safety factors to obtain an acceptable safe concentration of H_2S in the air should include consideration of the following important information:

1. The characteristic "rotten egg" odor of the gas can be detected at concentrations below those at which toxic effects have been observed;

2. Hydrogen sulfide is rapidly detoxified in the body, and the gas and it's known toxic effects do not appear to accumulate upon continued low exposures (e.g., below 10 ppm);

3. The toxic effects of H_2S appear to be determined primarily by the concentration of the gas in the air (or amount inhaled) rather than the time over which exposure to the gas occurs;

4. There is no direct evidence to indicate that children are more susceptible than adults to the toxic effects of H_2S. However, it is reasonable to assume that prenatal and early postnatal exposure to the gas should be restricted;

5. Differences among species in the known potency with which H_2S produces toxicity are not prominent. However, rats and mice appear to show higher sensitivity to nasal lesions and this may be attributed to anatomical characteristics of the rodent nasal passages; and

6. The application of certain safety factors and other factors to modify the NOAEL as conducted by the USEPA in calculation of the Reference Concentration for H_2S appear to lack scientific support given the known characteristics of the toxicity produced by the gas.

Health Effects of Low-Level

Hydrogen Sulfide in Ambient Air

(A Science Report to Governor John Engler)

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Introduction

Hydrogen sulfide (H_2S) is a colorless, highly toxic gas at high concentrations. It has a distinctive "rotten egg" odor and is produced by a variety of sources, including wastewater treatment plants, agricultural operations, paper mills and other manufacturing processes, and oil and gas development. Within Michigan, there is particular concern over H_2S associated with the exploration, production, and transportation of crude oil and natural gas. Some of these operations occur in areas of relatively dense residential development, and oil and gas operations have the potential to release H_2S in higher volumes than many other potential sources. Hydrogen sulfide gas has been produced in the state since the 1930's from various oil and gas reservoirs (MDEQ, 1998).

An extensive body of rules covering H_2S-containing wells, storage and treatment facilities, and pipelines was promulgated in Michigan in 1987 (MDEQ, 1998). Among other things, the rules prohibit nuisance odors from H_2S and prescribe installation of specific control equipment on H_2S-producing wells. Recently, however, public concern over H_2S associated with the oil and gas industry has escalated. In 1996, there was an intentional release (below worker safety standards) at a well just north of Manistee, Michigan that affected 11 citizens at businesses in the area. Also, construction of a pipeline to transport H_2S-bearing natural gas from a well in Oceana County to a processing plant in Manistee County raised fears among citizens concerning the possibility of harm from accidental releases. More recently, there has been controversy over the potential of H_2S release in a proposed well in the city of Farmington Hills, Michigan.

In response to these and other exposure and health concerns, the state has taken a number of actions, including: the issuance of administrative rules that prohibit the release of H_2S gas to the environment; improving the quality and distribution of information to citizens and local government on potential new H_2S wells; working more closely with local emergency response personnel in terms of acute exposure to H_2S; and meeting with stakeholder groups to identify health concerns and issues, and to formulate responses that could then be implemented as administrative rules.

Charge to the MESB

A considerable body of literature exists on the human health impacts resulting from exposure to high levels of H_2S. In contrast, very little data have been compiled regarding health impacts of much lower levels of the gas. On May 27, 1998, the Michigan Environmental Science Board (MESB) was charged by Governor John Engler (Engler, 1998) to review the available H_2S low-level data and to recommend a range of exposures that could be considered safe to human health (Appendix 1).

MESB Response

On July 9, 1998, a H_2S Investigation Panel (Panel), composed of five MESB members was convened to begin the investigation. The investigation consisted of the accumulation and evaluation of peer-reviewed and some non peer-reviewed literature and data on the subject. In addition, verbal and written testimony was considered at

four meetings (Harrison, 2000; 1998a; 1998b;1998c). The Panel, upon completion of the information gathering phase in the beginning of 1999, waited for completion of a study performed at the Chemical Industry Institute of Toxicology in which rats were exposed to low levels of H_2S for a period of 70 days. Results of those experiments were considered important to the successful completion of the task assigned to the MESB. Those results became available only recently.

Properties of Hydrogen Sulfide

Hydrogen sulfide is a flammable, colorless gas with a pungent odor characteristic of rotten eggs, and it is corrosive to certain metals. At a concentration of 300 parts per billion (ppb), the objectionable odor is distinct. The maximum concentration below which nearly all individuals could be exposed for up to one hour without perceiving a clearly defined objectionable odor is believed to be 100 ppb. However, some sensitive individuals can detect the presence of H_2S as low as 3 ppb (AIHA, 1991). Because H_2S is slightly heavier (1.19 times) than air, there is a potential for accumulation in pits, sewers, etc. under stagnant atmospheric conditions.

Sources of Hydrogen Sulfide

Natural Sources. The natural sources of H_2S are ubiquitous. Bacterial decomposition of proteinaceous material produces H_2S (Sittig, 1975). As a result, it is produced in many soils, coastal lagoons, tidal flats, swamps, salt and freshwater marshes, mudflats, and near-surface ocean waters (Warneck, 1988). Although H_2S is formed under both aerobic and anaerobic conditions, some of the H_2S will be oxidized to sulfates by microorganisms in an aerobic environment (Warneck, 1988). In the saturated or flooded soils, the highest fluxes of H_2S generally occur at night and in the early morning as the oxygen content in stagnant waters reaches a minimum. Hydrogen sulfide also occurs naturally as a component of natural gas, petroleum, volcanic gases, sulfur deposits and sulfur springs (Sittig, 1975).

Anthropogenic Emission Sources. Numerous man-made activities can release H_2S into the atmosphere. These include: the extraction, transport, and processing of natural gas, the extraction and refining of petroleum products, kraft mills, coke ovens, coal mining, foundries, some chemical operations, livestock production facilities, animal processing plants, tanneries, waste-water treatment plants, sewers, sanitary landfills and some water treatment plants. Table 1 presents a listing of 74 occupations that have the potential of H_2S emission exposure. Descriptions of these sources as well as typical emission factors can be found in Sittig (1975).

Since H_2S naturally occurs in natural gas and petroleum, any operation involving the extraction, transport or processing of these materials has the potential to release H_2S to the atmosphere. The H_2S content of natural gas varies from deposit to deposit and in Michigan ranges from zero to 63 percent (630,000 parts per million - ppm) (Vugrinovich, 1998). If the H_2S content of the gas is 300 ppm or more, the wells are subject to setback regulations and placed in one of four classifications depending upon H_2S concentration and potential maximum gas flow. Based on traditional dispersion modeling, the 100-ppm radius of exposure is calculated for a worst-case accident

scenario. The 100-ppm radius of exposure determines the required setback. The four classifications are: I - more than 300 feet, II - 100 to 300 feet, III - 30 to 100 feet, and IV - less than 30 feet. At present, the number of wells in Michigan in these four classes are: Class I, 152; Class II, 83; Class III, 216; and Class IV, 890 (Vugrinovich, 1998).

Table 1. Occupations with potential hydrogen sulfide exposure.

Animal fat and oil processors	Lithographers
Animal manure removers	Lithopone makers
Artificial-flavor makers	Livestock farmers
Asphalt storage workers	Manhole and trench workers
Barium carbonate makers	Metallurgists
Barium salt makers	Miners
Blast furnace workers	Natural gas production and processing workers
Brewery workers	Painters using polysulfide caulking compounds
Bromide-brine workers	Papermakers
Cable splicers	Petroleum production and refinery workers
Caisson workers	Phosphate purifiers
Carbon disulfide makers	Photoengravers
Cellophane makers	Pipeline maintenance workers
Chemical laboratory workers, teachers, and students	Pyrite burners
Cistern cleaners	Rayon makers
Citrus root fumigators	Refrigerant makers
Coal gasification workers	Rubber and plastics processors
Coke oven workers	Septic tank cleaners
Copper-ore sulfidizers	Sewage treatment plant workers
Depilatory makers	Sewer workers
Dyemakers	Sheepdippers
Excavators	Silk makers
Felt makers	Slaughterhouse workers
Fermentation process workers	Smelting workers
Fertilizer makers	Soapmakers
Fishing and fish-processing workers	Sugar beet and cane processors
Fur dressers	Sulfur spa workers
Geothermal-power drilling and production workers	Sulfur products processors
Gluemakers	Synthetic-fiber makers
Gold-ore workers	Tank gagers
Heavy-metal precipitators	Tannery workers
Heavy-water manufacturers	Textiles printers
Hydrochloric acid purifiers	Thiophene makers
Hydrogen sulfide production and sales workers	Tunnel workers
Landfill workers	Well diggers and cleaners
Lead ore sulfidizers	Woolpullers
Lead removers	

Source: NIOSH, 1977.

Ambient Concentrations of Hydrogen Sulfide

As a result of ubiquitous natural sources, background concentrations over continental regions average about 0.1 ppb. Higher concentrations, 1 to 4 ppb, have been reported over tidal flats, marshes and anaerobic soils. Concentrations over remote oceanic areas have been reported in the 0.01 to 0.02 ppb range (Warneck, 1988; Graedel, Hawkins and Claxton, 1986).

Areas that contain one or more of the man-made sources listed in Table 1 are likely to experience H_2S concentrations that exceed the continental background of 0.1 ppb. Unfortunately, urban H_2S measurements are extremely limited but indicate considerable variations in H_2S concentrations. The range of reported concentrations is from background up to about 50 ppb.

In Michigan, hourly total reduced gaseous sulfur (TRS) data were collected in Alma downwind of a petroleum refinery complex over 700 days from 1991 through 1994. The TRS will be an upper limit estimate of the H_2S as H_2S is generally the most abundant component of TRS. The monitoring was initiated because of citizen odor complaints. Most of the hourly values were at or below the minimum analytical instrument detection level of 1 ppb and the arithmetic mean for the four years was about 1 ppb. Hourly concentrations above 10 ppb occurred on only five percent of the days and accounted for less than 0.4 percent of the hourly readings. The maximum observed hourly concentration was 44 ppb (MDEQ, 1998).

Fate of Hydrogen Sulfide in the Atmosphere

Once emitted into the atmosphere, the principal removal mechanism of H_2S is the reaction with the hydroxyl ($\cdot OH$) radical (Warneck, 1988). The $\cdot OH$ radical is ubiquitous as it is formed in both pristine and polluted atmospheres via photochemically initiated reactions. In pristine atmospheres, water vapor is the precursor of $\cdot OH$ while in polluted atmospheres, volatile organic compounds (VOCs) serve as the main precursors. The removal reaction is:

$$H_2S + \cdot OH \rightarrow H_2O + HS$$

This is then followed by:

$$HS + 2O_2 \rightarrow SO_2 + HO_2$$

Based on the kinetics of these reactions, the globally averaged lifetime of H_2S in the atmosphere is about four days. During the daytime, during the summer, and in the presence of VOCs, the lifetime will be considerably shorter because $\cdot OH$ concentrations will be higher.

Hydrogen Sulfide Standards Issues

Regulatory challenges associated with the development of H_2S standards include whether to regulate H_2S based on nuisance, such as odor, or based on health effects (Pour, 1998). A survey of existing state H_2S standards was conducted the AERO Engineering Services (1998). Based on that survey, it was found that 20 states have developed H_2S standards. Of these, seven used odor-based threshold as the starting point for the development of its standard. The remaining 13 states used one of the following standard development approaches (Barrett, 2000):

1. Use of the U.S. Environmental Protection Agency's (USEPA) Integrated Risk

System (IRIS) as the basis;
2. Use of monitoring data to determine reasonable background values;
3. Use of the American Conference of Governmental and Industrial Hygienists' Threshold Limit Value as the basis; and
4. Use of health-based toxicological studies as the basis.

Few states have attempted to develop H_2S standards using health-based toxicological studies as its basis. An exception to this is Nebraska. In May 1997, the Nebraska Department of Environmental Quality (NDEQ) proposed the establishment of the following ambient air quality standards for total reduced sulfur (TRS) (NDEQ, 1997):

1. TRS = 10 ppm, one minute average;
2. TRS = 0.10 ppm, 30 minute rolling average;
3. TRS = 0.10 ppm, 30-day rolling average when the average relative humidity in 60 percent or less; and
4. TRS = 0.005 ppm, 30-day rolling average when the average relative humidity is greater than 60 percent.

The ambient air quality standards developed in Nebraska used total reduced sulfur compounds because H_2S was too difficult to measure separately. The standards were based on the National Institute for Occupational Safety and Health recommendation of 100 ppm for a healthy worker with a safety factor of ten added to make it safe for the general population (Pour, 1998).

In April 1998, the District Court of Lancaster County issued a temporary injunction prohibiting the enforcement of the resulting regulation. Although the Court concluded that the temporary injunction was "… *not likely to result in harm to the public* …'' (Witthoff, 1998), it also commented on the relationship between the standards proposed by the NDEQ and its ability to monitor them. The Court noted that the previously cited NDEQ proposed standards had been modified by Nebraska's Environmental Quality Council (NEQC) to exempt a "… *variety of industries and entities* …" known to emit TRS and, more relevant to this discussion, to "… *lower the concentration level of one of the standards by half while decreasing the exposure duration from one minute to instantaneous."* The NEQC had changed the proposed 10-ppm, one-minute average standard to 5 ppm, "instantaneous" at the request of Dakota City residents. During testimony, the state toxicologist advised the Court that its monitoring equipment could not measure any "instantaneous" reading. As a result, the Court held that "… *the inability to measure 'instantaneous' readings makes it impossible for NDEQ to determine violations of its own standard and impossible for those being regulated to determine whether they are or are not in compliance."* These events suggest that any proposed air standards should be measurable and routinely monitored by commercially available instruments.

Effective January 10, 1999, the NDEQ regulations specified the following method for TRS as follows (NDEQ, 1999):

> " *…levels of TRS in the ambient air shall be measured using a TRS thermal converter in conjunction with an SO_2 monitor. The SO_2 monitor shall be*

designated as an EPA reference method or equivalent method in accordance with 40CFR Part 50."

The regulation also explicitly described minimum specifications for the SO_2 monitor; i.e.,

1. Lower detection limit of 0.5 ppb;
2. Zero Drift less than 0.5 ppb in 24 hours and less than 1 ppb in 7 days at constant conditions;
3. Span Drift of less than 0.5 percent of the reading in 24 hours and less than 1 percent of the reading in 7 days at constant conditions;
4. Precision of 0.5 percent of the reading; and
5. Linearity of 1 percent of full scale.

The regulation states that the standards are met when both of the following conditions are met:

1. The one minute concentration is less than or equal to 10.0 ppm, and
2. The 30-minute rolling average is less than or equal to 0.10 ppm

Monitoring Limitations

Benner and Stedman's (1990) evaluation of available commercial measurement of sulfur gases indicated that reliable measurement at the sub-parts per billion level is not available. Tarver and Dosgupta (1995) have reported the development of an automated instrument that can continuously measure low levels of H_2S (detection limit 0.2 ppb for a sampling time of 2.5 minutes) for use in oil field operations. The Arizona Instrument Corporation's Jerome 631-X Hydrogen Sulfide Analyzer was employed in a screening study by the Colorado Department of Public Health and the Environment (CDPHE, 1998). The minimum time period over which the Jerome analyzer can provide averages is one minute, the limiting time period for most instruments. The response of the instrument is 1 to 50 ppm. Special instruments of the type described above are required for monitoring H_2S in ppb range and sub-parts per billion range. The detection limit of monitors currently in use by industry and the Michigan Department of Environmental Quality (MDEQ) is typically 1 ppm (Baker, 1998).

Monitoring Database for Hydrogen Sulfide Exposure in Michigan

Within Michigan, complaints regarding H_2S odors and possible health concerns are reported to the MDEQ. Since March 1979, a total of 469 complaints have been received and investigated (Harrold, 2000; Thomas, 1998). The majority of these complaints regarded odors; however, three were strictly noise complaints, two regarded fire or excessive flames, and one was a report of a vapor cloud.

The 469 complaints came from 31 different counties in Michigan. Of the 469 reports, ambient air H_2S testing was conducted in association with 67 complaints. These tests were completed at a variety of locations including at the well site, downwind and at the complainant's residence. The levels of H_2S detected ranged from 0 ppm (26 cases) to one report of 10,000 ppm, which was detected at a wellhead.

In addition to odor, 75 complaints listed associated health effects. These included 23 reports of nausea, 32 of headaches, and nine of respiratory problems. There were also five reports of eye irritation, one of skin irritation, one of insomnia, two of taste, and 11 of miscellaneous complaints. None of the 75 reports was followed up with a medical evaluation. Only seven of the 75 reports that listed health effects also had documented H_2S ambient air testing. Hydrogen sulfide levels in these seven cases ranged from 0 ppm to 10 ppm. Too little information exists to draw any conclusions from the available Michigan H_2S exposure data, the major deficiency being that the complaint-associated information lacks medical follow-up and that it is self-initiated.

Hydrogen Sulfide Health Effects

A complete and current review of the scientific literature regarding the adverse health effects from exposure of humans and laboratory animals to H_2S is available (ATSDR, 1999). The MESB Panel has also obtained copies of original reports published in the peer-reviewed literature and also is in possession of information that has not been published. This section of the MESB report on H_2S contains information derived from scientific reports pertaining to the Panel's task of identifying a level of H_2S in the ambient air that would not be considered harmful to public health when exposure occurred on a daily basis. Such exposure would occur to persons living downwind from industrial (e.g., petrochemical) or agricultural (manure holding) facilities or natural sources of the gas such as geological vents. Reports and information cited are those deemed relevant to the charge given to the Panel.

Lethal and High Exposures. Human exposure to H_2S in high concentrations (e.g., >500 ppm) causes a rapid progression of clinical symptoms that lead to death in a short period of time (minutes to a few hours). Exposure to concentrations that may cause death is almost always due to accidental inhalation and is not rare. The National Poison Control Centers Data Collection System reports 5,563 exposures during a 10-year period (1983 - 1992) with at least 29 deaths (Snyder *et al.,* 1995). Death-producing exposures usually occur in confined spaces such as sewers, manure pits, etc. After a few breaths in such atmospheres, the exposed person rapidly becomes unconscious (termed "knockdown") and death occurs presumably from respiratory arrest caused by dysfunction or death of cells in the area of the brain that sustains breathing.

Complete recovery from a high exposure episode has often been reported; however, in some cases long term and even permanent harmful effects remain (Kilburn, 1999). These persistent effects are often neurological or respiratory in nature and may have been caused by an essentially hypoxic (low oxygen) condition existing in persons who are unconscious from a high exposure to H_2S. Because a loss of oxygen utilization in tissues, particularly the brain, occurs in such poisonings it is possible to attribute persistent neuronal damage to this effect (Dorman, Brenneman and Struve, 1999; Milby and Baselt, 1999). Persistent damage is commonly observed clinically when brain tissues have been deprived of oxygen due to inadequate delivery of the gas or to interrupted utilization of oxygen by cells as is the case with H_2S poisoning. Thus, the

complete recovery of persons suffering from the clinical effects of H_2S exposure at moderate to high concentrations although reported, can be questioned (Milby and Baselt, 1999). Adequately documented reports of complete recovery remain scarce because rigorous methodology to detect abnormalities in apparently recovered patients is seldom applied, and confounding factors are often involved in cases of acute poisoning.

Dose-Response Relationships and Mechanism of Toxicity. Observations of effects in persons exposed to different concentrations of H_2S in the air indicate that there is a progression in the severity of adverse health effects. Because of the sensitivity of the olfactory nerves, it is possible for the human to detect the presence of H_2S in the air at a concentration of <0.1 ppm (Kilburn, 1999,). The odor is offensive at 3 - 5 ppm and at a concentration of 150 ppm the olfactory nerves are unable to detect the odor presumably because of olfactory nerve damage or sensory overload. Thus, odor is a sensitive marker of low exposure to the gas. At the detectable odor level of 0.01 ppm (Roth, 1999), adverse health effects directly attributable to H_2S have not been reported.

A dose dependent progression of adverse effects starting with eye irritation at 10 - 20 ppm and ending with collapse and death at 1000 - 2000 ppm is shown in Table 2. The primary mechanism for the toxic action of H_2S is a direct inhibition of the mitochondrial respiratory chain in cells by inhibiting the enzyme cytochrome oxidase (Kahn *et al.,* 1990; Lopez *et al.,* 1987; Nicholls *et al.,* 1976). Inhibition of this enzyme reduces the oxygen dependent metabolism of the cell, reduces cell energy sources (e.g., ATP), increases products of anaerobic metabolism such as lactic acid and produces cell death. Cells with a high oxygen demand such as those in brain and cardiac tissue are thought to be more sensitive to disruption of oxidative metabolism and may be considered selected targets for the toxicity of H_2S (Ammann, 1986).

Almost all of the observed effects produced in mammals poisoned with the gas may be attributed to the cellular anoxia produced by inhibition of cytochrome oxidase. However, other direct effects of H_2S have been suggested as contributors to its toxicity (for reviews, see ATSDR, 1999; Milby and Baselt, 1999; Roth, 1999). The relative contributions of cellular hypoxia resulting from inhibition of cytochrome oxidase and other direct effects of H_2S remain controversial. It seems likely that certain of the effects seen after high exposures are the result of alterations other than inhibition of the cellular respiratory chain and it is possible that these could account for as yet undiscovered effects from continual exposures to the gas that may occur in ambient air. Most current reviews of the scientific literature on H_2S toxicity do not fail to conclude that there is a lack of information regarding effects from low, long-term exposures that might be experienced from living near industrial or natural sources of the gas (Hessel and Melenka, 1999).

Table 2. Concentration response relationships for hydrogen sulfide.[a]

Effects	Hydrogen Sulfide ppm in air
Detectable Odor	0.008 - 0.1
Offensive Odor, Headache	0.25 - 0.30
Very Offensive Odor	3 – 5
ACGIH Limit	10
Occular Irritation/Conjunctivitis	20 - 100
Rapid Loss of Sense of Smell	150 - 250
Sudden Loss of Consciousness	500 - 1000
Collapse, Apnea, Death	1000 - 2000

a. Adapted from Milby and Baselt, 1999.

The detoxification of H_2S after it is inhaled is rapid and occurs in the blood and most other tissues of the body. Hemoglobin in the blood and metalloproteins in other tissues participate in the oxidation of H_2S to thiosulfate and ultimately sulfate which appears in the urine. These products apparently do not inhibit cytochrome oxidase (Beauchamp *et al.*,1984). Small amounts of H_2S are excreted unchanged in the expired air. As the concentration of H_2S increases in blood and other tissues due to exposure, the processes of detoxification to sulfates become increasingly less effective. This leads to an accumulation of H_2S in tissues and if it is sufficiently high, toxicity is observed. Thus, H_2S has a threshold concentration in the air for producing toxicity and the gas is not considered a cumulative toxin when it is present in the air at low concentrations (Bhambhani, 1999; Beauchamp *et al.*, 1984). In contrast, Roth (1999) presents the view that there is evidence to suggest it is a cumulative toxin. While this important point is controversial, the studies suggesting cumulative toxicity are primarily a result of repeated moderate to high exposures of H_2S that may produce small increments of hypoxia-induced neuronal cell death. This effect is not likely to occur at low concentrations of the gas associated with environmental exposures as these are rapidly detoxified.

Examination of the Effects of Low Concentrations of Hydrogen Sulfide. The focus of the charge given to the MESB by the Governor (Appendix 1) is to estimate from available information the concentration of H_2S in air that will not present a health threat to persons living in conditions in which there is daily exposure to low concentrations of the gas in air. Not considered relevant in this context are results from studies of the effects of acute high exposures leading to toxicity and permanent damage emanating from a cellular anoxic condition. A recent review of harmful effects in persons exposed to the gas occupationally and accidentally suggested that complete recovery is not always observed, even in persons who did not exhibit unconsciousness (Kilburn, 1999). A lack of full recovery was used to support the premise that lower concentrations of H_2S

may be expected to produce harmful effects. While this seems a reasonable suggestion, the need for identifying the threshold for observing the toxic effects of H_2S remains. The notion that there is an exposure level to H_2S that is not harmful is reasonable because this substance is a product of normal body processes, is found in tissues, and some evidence exists that it is a necessary component for certain types of nerve transmission (Kimura, 2000).

Community Studies. Data reported from studies of communities experiencing low exposures to H_2S and other contaminants from nearby industrial or natural sources could provide an indication of a threshold for adverse health effects. Unfortunately, these studies usually provide equivocal results because of the mixture of pollutants in the air, which usually includes organic sulfides, sulfur dioxide and particulates. Exposure to pulp mill emissions in two communities in Finland indicated that self-reported respiratory symptoms (wheezing, shortness of breath) and eye irritation/conjunctivitis were higher in the exposed communities compared to a third community with no exposures (Jaakkola *et al.,* 1990). Children in the same communities did not appear to be more sensitive to the effects of exposure when compared to adults (Marttila *et al.,* 1994). A subsequent study in which measured exposure levels were used indicated that average 24 hour exposures of <0.007 ppm H_2S (peak 4 hour exposure was 0.095 ppm) H_2S exhibited no significant respiratory or ocular effects, but above that level respiratory symptoms were observed according to information obtained from questionnaires (Marttila *et al.,* 1995). These studies and others like them, although informative, cannot be given great weight in identifying the threshold exposure for toxicity from H_2S. The reason for this includes the use of self-reporting in the assessment of health outcomes and the fact that other air pollutants undoubtedly contributed to the reported effects. At best, the information suggests that H_2S may contribute to respiratory symptoms when mixed with other air pollutants originating from pulp production.

Controlled Human Exposures. A series of experiments was performed by Bhambhani and coworkers (Bhambhani *et al.,* 1996a; 1996b; 1994; Bhambhani and Singh, 1991) to examine the dose-related effects of low exposures of H_2S on exercising volunteers. The use of exercise increased the exposure to H_2S by raising the rate of breathing, thereby increasing the detection of increasing anaerobic metabolism induced by the exposure and approximating the exposure situation of an exercising worker. In the first series of experiments, 16 healthy, male volunteers undertook increasing increments of bicycle exercise while inhaling 0 (control), 0.5, 2.0 and 5.0 ppm of H_2S, on separate occasions, while multiple physiological measurements were made (Bhambhani and Singh, 1991). The results indicated that there were no significant changes in the cardiorespiratory processes at any exposure level and at any exercise level. Heart rate, ventilation rate, oxygen uptake, and carbon dioxide production were unaltered. However, blood lactate increased significantly during exposure to 5.0 ppm H_2S. These results indicated that anaerobic metabolism is increased by the presence of the sulfide but whether or not this is due to inhibition of cytochrome oxidase cannot be determined from the results.

A second series of experiments employed 13 male and 12 female healthy volunteers exposed to 0 (control) and 5.0 ppm of H_2S while exercising for 30 minutes (Bhambhani *et al.,* 1996a; 1994). In all volunteers, the 5.0-ppm exposure did not change arterial blood gases, hemoglobin saturation levels, or cardiovascular and metabolic responses. Statistically, citrate synthetase, a marker of aerobic metabolism, was the only enzyme to show a decrease in activity. Lactate dehydrogenase and cytochrome oxidase were not altered in muscle tissue taken by biopsy. The results confirmed those from the previous study with the exception that lactate levels in blood were slightly higher but not statistically different in the exposed individuals. Both healthy, exercising men and women showed little response to H_2S.

Another study using nine males and 10 females showed that healthy persons exposed to 10 ppm H_2S for 15 minutes while exercising showed no changes from control in a series of respiratory measurements (Bhambhani *et al.,* 1996b). A final study in this series employed 28 healthy volunteers exposed to 0 (control) or 10 ppm of H_2S while exercising for 30 minutes. The only significant treatment-related effects observed in the battery of measurements were an increase in muscle lactate in men and women, a decrease in muscle cytochrome oxidase in men and an increase in this enzyme in women.

In all of the studies performed in this series, none of the volunteers reported adverse health effects subsequent to H_2S exposure. Taken together, the results of these studies do not indicate changes take place in healthy adults that signal the initiation of a toxic response from H_2S exposure up to 10 ppm. The magnitude of the few exposure-related changes that were observed, the sporadic occurrence of the changes and the lack of a functional change in the cardiorespiratory system are not consistent with a toxic response. Rather, they may be reasonably classified as a marker of exposure to the gas.

There are limitations to the controlled studies presented above, and they have been pointed out by the investigator (Bhambhani, 1999). The H_2S exposure methodology used in all the experiments protected the exercising volunteers from exposure to the nose and eyes. Thus, they could not smell the gas and were not subject to eye irritation, both are sensitive outcomes connected with H_2S exposures. Because the volunteers were physically fit and healthy, the results cannot easily be applied to the sedentary and unhealthy portion of the population. In addition, the volunteers did not have previous exposures to the concentrations of H_2S used in the experiments. If cumulative toxicity is involved in continuous exposure to the gas, it was not mimicked in these experiments.

Jappinen *et al.* (1990) exposed a group of ten asthmatic subjects (3 men, mean age 40.7 years and 7 women, mean age 44 years) to 2 ppm H_2S for 30 minutes in a closed chamber. Patients with severe asthma were excluded because the subjects were to be withdrawn from medication for two days prior to the experiments. Respiratory function was measured before and after exposure to the gas. All subjects experienced unpleasant odor at the start of the exposure but rapidly became accustomed to it and

three of the subjects experienced headache after exposure was completed. There were no changes in forced vital capacity (FVC), forced expiratory volume in one second (FEV_1) and forced expiratory flow (FEF) as a result of exposure. Calculated airway resistance was slightly decreased in two and increased in eight subjects with no significant change in mean values and no clinical symptoms were noted. Calculated specific airway conductance was decreased in six and increased in four subjects showing no significant change in mean values. In two of the individuals there was a change of over 30 percent in both of these calculated values, which is indicative of bronchial obstruction. The authors suggested that this effect might be due to the irritant nature of H_2S. They also caution that because of the low number of subjects studied, the results from this experiment should be considered preliminary.

In the same report Jappinen *et al*. (1990) describe the results of respiratory measurements made on 26 male pulp mill workers who were exposed occupationally to H_2S in the range of 2 ppm to 7 ppm. Respiratory function (FVC and FEV_1) and bronchial responsiveness to histamine challenge of the workers was measured after a day of not working and again after working for a day in which exposure to H_2S took place. No significant changes in respiratory function or bronchial responsiveness related to exposure to H_2S were found. The values of the respiratory function tests of the workers prior to and after exposure were not significantly different when compared to published values derived from the Finnish general population.

Animal Studies. A number of published reports exist concerning the toxicity of H_2S in laboratory animals. The information in those reports generally indicate that toxic effects observed in animals exposed by inhalation of the gas in high concentrations are identical to those observed in humans exhibiting acute toxicity to the gas. Compilation of the results obtained from use of concentrations of H_2S that produce serious systemic toxic effects and death in humans and laboratory animals indicate that species differences in toxicity are not prominent (ATSDR, 1999). Concentrations that produce effects in the respiratory, cardiovascular, and nervous systems are similar (i.e., less than an order of magnitude) in rats and mice and humans. Comparison among species of result from experiments using concentrations that produce less serious symptoms (e.g., headache, dry mouth) in humans are difficult because of obvious differences in the way the effect can be detected. The mechanism of action to produce serious effects appears to be the same in each species as cellular respiration and energy production controlled by cytochrome oxidase in the mitochondria is inhibited by H_2S in all species tested. This mechanism is consistent with effects observed in multiple organ systems in laboratory animals given acute, high exposures.

The reversibility of the toxic effects initiated by cytochrome oxidase inhibition by H_2S observed in many clinical cases has been demonstrated in laboratory animals. The enzyme in the lung of rats was found to be inhibited by 15 percent after four hours of exposure to 50 ppm and 43 percent after 200 ppm. Activity returned to normal after cessation of exposure to these concentrations (Kahn *et al.,* 1990). Changes in discrimination avoidance behavior of rats exposed to concentrations of H_2S at 100 ppm for one hour returned to normal within an hour after a two-hour exposure. (Higuchi and

Fukamachi, 1977).

It is important to note that the concentration of irritant gasses such as H_2S is much more important in the determination of toxic outcome (LD_{50}) than is the duration of exposure (ten Berge, Zwart and Appelman, 1986). The concentration-exposure duration relationship for a toxic effect of these gasses follows the relationship $(C^n)(t)=k$, where *C* equals concentration, *n* is usually between 1.0 to 3.5, and *k* is a constant. This relationship indicates that an increase in exposure duration will usually have a relatively small effect on the toxic outcome when compared to the effect resulting from an increase in concentration. It has been observed that the product of dose and duration is less predictive of H_2S toxicity in humans than the magnitude of the dose (Kilburn, 1999). This suggests that cumulative toxicity upon continued low exposure to H_2S appears to be unlikely because of rapid detoxification and reversibility of adverse biological actions.

Chronic exposure of rats and mice to H_2S six hours/day, five days/week for 90 days using concentrations in the air of 0 (control) 10, 30 and 80 ppm of the gas showed no increase over control in mortality (Morgan *et al.,* 1983a; 1983b; 1983c). In these studies, no histopathological effects were noted in any of the organ systems examined including respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, endocrine, dermal, immune, and ocular. Small changes in body weight at the end of the study were noted in some treatment groups. No changes in metabolic parameters and serum enzymes were noted. The lack of detectable effect on the structure and function of major organ systems indicated that cumulative toxicity in specific cells and tissues were not occurring at the doses used. However, the histological data from this study are being re-evaluated at this time and the study is not completed. This was thought to be important (Dorman, 2000) because of the lesions observed in olfactory mucosa in the study described below.

The sensitivity of the olfactory system to low concentrations of H_2S and previous reports of acute necrosis of olfactory mucosae in rats exposed to high concentrations of the gas prompted a recently reported study of the effects of long-term, low dose exposure to cells in the rat nasal cavity (Brenneman *et al.,* 2000). Animals were exposed to 0 (control), 10, 30 or 80 ppm, six hours/day for 10 weeks. Olfactory nerve cell loss occurred in selective portions of the nasal cavity in a concentration dependent manner at 30 and 80 ppm. Basal cell hyperplasia, a regenerative response secondary to death of the nerve cells, was also observed. The damaged sites were mapped and compared to modeled airflows in the rat nasal cavity. The toxic effects were observed in caudal areas that have a high surface area and slower air flow, which increased localized tissue exposure to the gas in that region of the nasal cavity. The localization of lesions to olfactory epithelium, even though nasal regions containing these cells are exposed only to an estimated 16.5 percent of the inhaled chemical dose suggests that these cells exhibit higher sensitivity to the toxic effects of the gas. In this relatively long-term toxicity study, the only effects observed in the treated animals were nasal lesions in animals receiving the higher concentrations. A no observable adverse effect level (NOAEL) was observed at 10 ppm.

The results of a reproductive and developmental neurotoxicity study of H_2S were recently reported by Dorman *et al.* (2000). This extensive study utilized exposure to 1, 10, 30 and 80 ppm of the gas in male and female Sprague Dawley rats. The treatment was initiated two weeks before mating and continued throughout pregnancy and for 18 days after birth of the pups (except for five days immediately after birth). The offspring were also exposed from day 5 through day 18 after birth. Adult, male rats were exposed for a total of 70 days and nonpregnant females for 24 days. Exposure to H_2S did not alter any parameter of reproductive function in the adult animals, including mating and fertility index, sperm number and quality. In the offspring, the litter size and growth parameters were normal. No evidence of teratological effects in the offspring occurred and there was no evidence of exposure-related changes in developmental landmarks in the offspring. Selected tissues were examined for histopathology in the adult animals (reproductive organs) and their offspring (brain) and none was found except for the nasal effects reported separately (Brenneman *et al.,* 2000). A behavioral testing battery was applied to the offspring and no exposure-related effects were found. Male rats exposed for 70 days did not show evidence of H_2S toxicity in a full battery of testing (Dorman, 2000).

The report of Dorman *et al.* (2000) failed to confirm effects observed in pregnant Sprague Dawley rats exposed to 75 ppm of H_2S by Hayden, Goeden and Roth (1990). These workers observed a slight increase in gestation length and effects on hair development in the offspring. Minor and possibly anomalous increases in liver cholesterol occurred in 14 day old pups but not in 7 or 21 day old offspring. Hannah and Roth (1991) indicated that exposure of pregnant rats to 20 ppm of H_2S throughout pregnancy and lactation interrupted normal dendritic growth of Purkinje cells in the brain of offspring examined 21 days after birth. A morphometric analysis of brain cell arborization was not conducted in the study reported by Dorman *et al.* (2000) so this effect has yet to be confirmed in another investigation. A review of neuronal toxicity of H_2S by Roth (1999) emphasizes that development of the brain is altered by exposure to the gas. Levels of several neurotransmitters including aspartate, glutamate, norepinephrine, and taurine were found to be altered in specific areas of the brain prior to weaning in the offspring of pregnant rats exposed to 75 ppm of H_2S. There was also evidence that some of the modest changes observed returned to normal in the young animals. Although behavioral testing has not indicated that alterations of brain neurotransmittters have a functional impact (Dorman *et al.,* 2000), further examination of the biochemical and functional aspects of brain development in H_2S exposed animals is warranted. Emphasis should be placed on examination of the concentration-response relationships involved.

Toxicity in Children. Neurotoxicants such as lead and methylmercury are known to be more potent in children than in adults. There are very little data available from laboratory animal experiments indicating that the young are more susceptible to the effects of H_2S exposure. However, a recent report indicated rats exposed *in utero* and from day 5 to day 18 after birth exhibited no evidence of toxicity or behavioral deficits with exposures up to 80 ppm (Dorman *et al.,* 2000). In contrast, (Roth, 1999) reported

alterations in brain development caused by exposure of pregnant rats to 75 ppm throughout gestation and lactation. This effect suggests that young animals may be more vulnerable to the neurotoxicity of the gas. Using existing information on H_2S toxicity, it is not possible to come to a reliable conclusion regarding the relative sensitivity of H_2S in adults and children.

Conclusions

Data pertinent to health risks of long-term exposure of humans and laboratory animals to H_2S in the air have been evaluated with the goal of identifying a safe (acceptable low health risk) level of the chemical in air. It is expected that the level chosen as safe will be protective of persons having higher sensitivity to the deleterious effects of H_2S than most segments of the population. Not considered in the sensitive group are individuals who exhibit extreme sensitivity because of an immune-based response (i.e., allergy) to the chemical. Such responses often do not exhibit typical dependence on the dose (or concentration) of the chemical to which extreme sensitivity has developed. A safe dose for these individuals could be below natural background levels.

Choosing a NOAEL. Because H_2S exposure has not been associated with a higher incidence of cancer in humans or laboratory animals (ASTDR, 1999), a concentration in the air considered as safe is likely to be calculated by the NOAEL safety factor method utilized by the USEPA. This is accomplished by dividing a chosen NOAEL by a safety factor. This essentially reduces the NOAEL value obtained from scientifically based observations to compensate for deficiencies in the data from which the NOAEL is derived. The ideal NOAEL would be obtained from valid measurements made in an adequate number of people exhibiting high susceptibility to the toxic effects of H_2S. These individuals would be exposed daily to air containing a known amount the gas over a lifetime. Appropriate measures would be taken to correct for confounding factors that may also impact the adverse health outcome presumably resulting from exposure to the gas. Ideal data from exposed humans do not exist for H_2S, and safety factors to reduce an experimentally determined NOAEL will be needed to increase the certainty of an estimated safe concentration in the air.

The selection of the safety factor value to be applied in the calculation of a health-based, acceptable air concentration represents a decision involving a high degree of social and public health policy. This statement is made knowing that a scientific basis for the application of safety factors or uncertainty factors has been attempted but remains deficient. The experimentally determined NOAEL represents a value that is grounded in scientific data and judgment and the goal of this report is to provide that value. Regulatory scientists and decision makers will presumably select safety factors to reduce that NOAEL to a value that has adequate certainty for protection of public health.

The NOAEL for H_2S in the ambient air appears to be from 2 ppm to 10 ppm. This conclusion is derived from an examination of published scientific data relevant to this determination. The studies considered relevant are shown in Table 3. From controlled

human exposures of a short duration, Bhambhani *et al.* (1996a; 1996b; 1994) and Bhambhani and Singh (1991) have found that sensitive measures of the effects of the gas on respiratory enzymes and function indicate that no consistent effect is observed at 5.0 ppm. In those studies, an increase (approximately 50%) was detected in blood lactate after a single 30-minute exposure of 10 ppm H_2S exposure. This is an effect to be expected in exercising adults because the subjects utilized in the study were undoubtedly undergoing an increase in lactate-producing anaerobic metabolism as a result of the exercise alone. Although a small increase in blood lactate occurred as a result of exposure, no changes in the target enzyme for H_2S toxicity (cytochrome oxidase) were observed after 10-ppm exposures in that series of studies. The major difficulty in utilizing the Bhambhani *et al* (1996a; 1996b; 1994) and Bhambhani and Singh (1991) studies for selection of the NOAEL is that they were conducted in healthy adults who were not exposed to low concentrations of H_2S on a daily basis.

The other study considered important to the determination of a NOAEL is that reported by Brenneman *et al.* (2000) who measured a loss of olfactory neurons in the nasal mucosa of rats exposed by inhalation for 70 days to H_2S at a level of 30 ppm. Prominent species differences in the anatomy of nasal passages exist between rodents and primates, including humans. These species differences may predispose the rat to higher H_2S tissue concentrations in the affected area. The olfactory neurons in the rat exhibit high sensitivity to H_2S as no abnormalities were observed in other organs or in important functions measured in the studies (e.g., reproduction, development, behavior) of animals exposed to concentrations of the gas as high as 80 ppm for 70 days.

The obvious difficulty with extrapolation of laboratory animal data to humans is possible differences between the rodent and human in responses to H_2S. To the Panel's knowledge, there are no data from valid measurements indicating that the human is an order of magnitude or more sensitive to the toxic effects of H_2S exposure than the rat or other animals used in the laboratory. Given the physical/chemical characteristics of the gas, such as its reactivity with tissue components and its favorable solubility in water, it would appear that at low exposures (i.e., low concentrations in the nasal cavity) it retains the characteristic of a Category 1 gas (USEPA, 1994). These types of gasses exhibit characteristic higher concentrations in the nasal region of the respiratory system at low air concentrations and deleterious effects would be regionalized to the upper respiratory system, primarily cells located in the nasal cavity. These types of gasses exhibit characteristic higher concentrations in the nasal region of the respiratory system at low exposure concentrations and deleterious effects produced are regionalized to the nasal mucosa. Further, it is known that other irritant gasses produce olfactory lesions similar to those produced by H_2S (Morgan, 1991).

Table 3. No Observable Adverse Effect Levels (NOAEL) and Lowest Observable Adverse Effect Levels (LOAEL) for hydrogen sulfide inhalation at low exposures.

Species	Exposure Description	NOAEL (ppm)	LOAEL (ppm)	Type of Measurement	Reference
Human	Controlled H_2S, 15-30 min. exercising adults	2.0	5.0	Metabolism, found increases in blood lactate	Bhambhani *et al.,* 1994-1996, and Bhambhani and Singh (1991)
Human	Controlled H_2S, 30 min. adult asthmatics	2.0	None	Respiratory	Jappinen *et al*., 1990
Human	Pulp mill workers (mixed sulfides)	2.0 - 7.0	None	Respiratory	Jappinen *et al*., 1990
Mouse	H_2S inhalation, 3 mo. toxicology study	30.0	80.0	Tox. battery, found nasal inflammation	Morgan *et al*., 1983a
Rat	H_2S inhalation, 90-day toxicology study	80.0	None	Tox. battery	Morgan *et al.,* 1983b; 1983c
Rat	H_2S inhalation, 3 mo. toxicology study	10.0	30.0	Tox. battery, found nasal lesions	Brenneman *et al.,* 2000
Rat	H_2S inhalation during pregnancy and lactation	80.0	None	Neuro-development and toxicity	Dorman *et al.,* 2000
Rat	H_2S inhalation during pregnancy and lactation	None	20.0	Development, changes in in Purkinje cells	Hannah and Roth 1991
Rat	H_2S inhalation during pregnancy	100.0	None	Development	Saillenfait, Bonnet, and deCeaurriz,1989

Regulatory Considerations. An examination of the basis for some current regulatory limits for H_2S provides additional perspective regarding an acceptable level of the gas in the ambient air. The Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Level (MRL) for H_2S inhalation utilizes a 1983 study in mice (Morgan *et al.,* 1983c) in which 90-day exposures to 80 ppm (lowest observable adverse effect level - LOAEL) produced nasal mucosa inflammation and 30 ppm (NOAEL) exposures produced no effects. The NOAEL was adjusted to 5.4 ppm to account for the fact that the mice were only exposed for six hours/day for five days instead of 24 hours/day for seven days. Next, a NOAEL human equivalent concentration ($NOAEL_{HEC}$) was calculated for an apparent “gas: respiratory” effect in the extra thoracic region (USEPA, 1994). This “correction” further reduced the $NOAEL_{HEC}$ to 0.81 ppm. An Uncertainty Factor (safety factor) of 30 was then applied to produce an MRL of 0.03 ppm (ATSDR, 1999).

The USEPA (1994) has calculated a Reference Concentration (RfC) for chronic inhalation of H_2S using the NOAEL of 30 ppm from the 90-day mouse exposure study conducted in 1983 (Morgan *et al.,* 1983c). Calculation of an exposure time adjustment and $NOAEL_{HEC}$ was made as in the MRL method described above. An Uncertainty Factor (safety factor) of 1000 was applied making the RfC 0.001 microgram/cubic meter (or 0.0007 ppm). This RfC value is below the odor detection limit of H_2S and is approximately four orders of magnitude below the threshold level (10 ppm) causing ocular irritation, one of the earliest, completely reversible effects in humans (Kilburn, 1999).

As briefly described above, prior to application of Uncertainty Factors to reduce a NOAEL obtained in laboratory animals to a value that would produce a safe air concentration (RfC or MRL in humans, two adjustments were are made in the NOAEL. These are referred to above as an adjustment for time of exposure and the $NOAEL_{HEC}$ calculation. Both of these adjustments reduce the experimentally based NOAEL and can be considered conservative (protective) adjustments. They are equivalent to applying an additional safety factor of 40. Both of these NOAEL adjustment procedures may be questioned in relation to their application to H_2S. The actual concentration - time relationship observed for that gas places more importance on peak concentrations than on the duration of exposure (Kilburn, 1999; ten Berge, Zwart and Appelman, 1986). A relatively greater importance for concentration of the gas rather than time of exposure is not consistent with the time adjustment applied to reduce the NOAEL obtained from a study using animals. The adjustment utilizing HEC implies that the rodent nasal mucosa is less sensitive than the human. This may not be the case as a recent report suggests that the rat may be more sensitive to H_2S than the human (Dorman, Brenneman and Struve, 1999). The reasons given for this are:

1. The rat is an obligatory nasal breather;
2. The rat has a much higher fraction of the olfactory mucosa-containing susceptible olfactory neurons; and
3. The airflow characteristics of the rat favor greater deposition of H_2S in certain nasal regions.

In addition, the commonly used HEC correction of animal NOAELs by (USEPA, 1994) apparently lacks validation and its use should be questioned according to recent data obtained from rats exposed to H_2S (Brenneman *et al.,* 2000; Dorman *et al.,* 2000).

The difference between the RfC value of 0.0007 ppm developed by the USEPA and the MRL value of 0.03 ppm developed by the ATSDR is large and the basis for this difference is unclear. Both values utilize the NOAEL obtained from the same 90-day exposure study in mice. Both calculated values were developed to protect human health from long-term exposure to H_2S in ambient air. The large difference results from the application of different Uncertainty Factors, which reduce an already lowered ("adjusted") NOAEL obtained from a study in mice. Justification for the use of higher Uncertainty Factors in the RfC calculation is provided in the USEPA's IRIS (USEPA,

1994) and includes the use of default procedures, uncertainty of the animal to human extrapolation in light of small changes in respiratory values in two out of 10 exposed asthmatic adults (Jappinen *et al.,* 1990), and a lack of application of sensitive measures of neuronal damage. Clearly, the data available from animal studies have increased with the emerging publication of new studies using 10-week exposures in rats (Brenneman *et al.,* 2000; Dorman *et al*., 2000; Dorman, Brenneman and Struve, 1999). In the absence of new human studies, the RfC calculation should utilize this information obtained using more advanced methodology (Morgan *et al.,* 1983a; 1983b; 1983c) along with justifiable safety factors.

There are a number of occupational regulations for H_2S that provide additional perspective for a safe level of the gas in the workplace. The U.S. Department of Labor's Occupational Safety and Health Administration indicates a maximum level in the workplace is 20 ppm and 50 ppm for a 10-minute exposure. The Centers for Disease Control and Prevention's National Institute of Occupation Safety and Health has a maximum level of 10 ppm over 10 minutes. The American Conference of Governmental Industrial Hygienists has a time weighted average limit of 10 ppm. Occupational limits are commonly set with the assumption of limited time exposures (e.g., the work day or less) in a healthy worker population and are usually higher (less conservative) values than that developed for the general population, which includes more susceptible individuals and children, and lifetime exposures.

Recommendations

The MESB was charged by Governor John Engler (Engler, 1998) to recommend a range of exposures to H_2S that could be considered safe to human health. Based on the scientific literature reviewed in this report, the NOAEL of H_2S in ambient air is between 2 and 10 ppm. The lower value is from studies showing no exposure-related increase in blood lactate, a marker for anaerobic metabolism, in exercising adults. This effect was observed after 5 ppm H_2S exposure for 20 minutes. It represents a physiological change that can result from inhibition of the enzyme cytochrome oxidase. Complete inhibition of that enzyme is responsible for the acute toxic effects of H_2S. No consistent effect on the enzyme itself could be detected in the individuals exposed to these low levels of the gas. The higher value, 10 ppm, was the NOAEL found in a recent 70-day exposure study using laboratory animals (rats). A concentration of 30 ppm of the gas produced evidence of nasal lesions affecting olfactory nerve cells in that study. This is a definite toxic effect in a species that may be more sensitive than the human to nasal lesions from irritant gasses such as H_2S.

NOAEL values from experiments of high quality are modified by safety factors to increase the certainty that humans exhibiting high sensitivity to the toxic effects of the gas will be protected. These safety factors should be applied with knowledge of the characteristics of the toxicity of H_2S gained from human and laboratory animal experiments. Selection of safety factors to obtain an acceptable safe concentration of H_2S in the air should include consideration of the following important information:

1. The characteristic “rotten egg” odor of the gas can be detected at concentrations below those at which toxic effects have been observed;

2. Hydrogen sulfide is rapidly detoxified in the body and the gas and it’s known toxic effects do not appear to accumulate upon continued low exposures (e.g., below 10 ppm);

3. The toxic effects of H_2S appear to be determined primarily by the concentration of the gas in the air (or amount inhaled) rather than the time over which exposure to the gas occurs;

4. There is no direct evidence to indicate that children are more susceptible than adults to the toxic effects of H_2S. However, it is reasonable to assume that prenatal and early postnatal exposure to the gas should be restricted;

5. Differences among species in the known potency with which H_2S produces toxicity are not prominent. However, rat and mice show higher sensitivity to nasal lesions and this may be attributed to anatomical characteristics of the rodent nasal passages; and

6. The application of certain safety factors and other factors to modify the NOAEL as conducted by the USEPA in calculation of the RfC for H_2S appear to lack scientific support given the known characteristics of the toxicity produced by the gas.

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Appendix

May 27, 1998 Correspondence to the Michigan Environmental Science Board from Governor John Engler

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STATE OF MICHIGAN
OFFICE OF THE GOVERNOR
LANSING

JOHN ENGLER
GOVERNOR

May 27, 1998

Dr. Lawrence Fischer
Chair
Michigan Environmental Science Board
300 South Washington Square
Suite 340
Lansing, Michigan 48933

Dear Dr. Fischer:

There has been recent concern expressed over health impacts of hydrogen sulfide in the atmosphere. I am requesting the assistance of the Michigan Environmental Science Board (MESB) in assessing this issue.

Hydrogen sulfide originates from a variety of sources, including oil and gas production, wastewater treatment plants, agricultural operations, and manufacturing processes. The acute effects of exposure to high levels of hydrogen sulfide are well-documented. There is more uncertainty and difference of opinion as to the effects of long-term exposure to low levels of hydrogen sulfide.

Specifically, I request that the MESB:

1. Review and evaluate available data and studies on the human health effects of hydrogen sulfide at low concentrations (i.e., at and below the occupational safety standards). The evaluation should include factors of hydrogen sulfide concentration in ambient air, duration of exposure, and variations in susceptibility to hydrogen sulfide among the general population.

2. Recommend a range of exposures that can be considered safe, taking into account variations in human susceptibility. The recommendation should be based on an evaluation of relative risk using time-weighted exposures to hydrogen sulfide among the general population.

RECYCLED PAPER

I am directing the Michigan Department of Environmental Quality (DEQ) to support the Board in facilitating this evaluation. Please provide the DEQ with the results of your evaluation as soon as possible.

Thank you for your continuing service to the citizens of Michigan.

Sincerely,

John Engler
Governor

JE/dkl/pw

cc: Russell Harding, Director, DEQ
Keith Harrison, MESB
Jim Haveman, DCH
Kathy Wilbur, CIS
John Strand, PSC

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